Maine Geological Survey
DEPARTMENT OF CONSERVATION
Walter A. Anderson, State Geologist

OPEN-FILE NO. 85-88

Title: Radon in the Domestic Environment and its Relationship to Cancer: An Epidemiological Study

Author: E. Melanie Lanctot

Date: 1985

Financial Support: This research was made possible by the Maine Geological Survey and by grants from the Maine State Legislature, Augusta; the Guy Gannett Foundation, Portland; the American Cancer Society, Maine Division, Brunswick; the Maine Cancer Research and Education Foundation, Portland; the Maine Department of Human Services, Augusta; the Maine Lung Association, Augusta; and the Maine Medical Center Cancer Research Fund, Portland.

Contents: 39 page report

RADON IN THE DOMESTIC ENVIRONMENT
AND ITS
RELATIONSHIP TO CANCER:
AN EPIDEMIOLOGICAL STUDY

A Master's thesis submitted to the
State University of New York at Stony Brook

by

E. Melanie Lanctot
Water Resource Analyst
Maine Geological Survey

---

with technical assistance
and support by

Dr. Peter W. Rand, Director of Research
Maine Medical Center, Portland

Dr. C. T. Hess, Professor of Physics
University of Maine, Orono

Dr. Gregory F. Bogdan, Epidemiologist
Maine Department of Human Services

Dr. Marc C. Loiselle, Senior Geologist
Maine Geological Survey

Published by the

Maine Geological Survey
DEPARTMENT OF CONSERVATION

Open-File Report 85-88

1985

ABSTRACT

The purpose of this study was to determine if the radon levels found in homes in Maine are associated with an increased incidence of cancer, especially cancer of the lung. To accomplish this, data were collected on three groups of patients: lung cancer patients, other cancer patients, and a control group of patients free of cancer. To be selected for the study, a patient had to be over 35 and to have lived in a house supplied by a drilled well for at least 10 years. Demographic information, including occupation, history of smoking, and medical history, was gathered through questionnaires. Data on the water supply was also requested in the questionnaire, and geological information was taken from bedrock and surficial maps in order to better predict where high radon levels might occur.

The metamorphic grade of the bedrock showed the only strong association with water radon levels, with the mean radon level tripling from low-grade to highly altered rock, and doubling from high-grade metamorphic rock to granite. No clear associations were observed between water radon levels and depth or yield of the well, or the type or thickness of the overburden.

Water radon, however, accounted for only 16% of the variance in air radon, suggesting that soil is a more important source of radon than was originally thought. Average air radon levels were highest in homes built on permeable sand and gravel and lowest on dense clays.

After confounding variables were compensated for by stratification, one category of patients evidenced a significantly higher risk of lung cancer. Men under the age of 65 who were exposed to over 3 pCi/l of airborne radon had 8.2 times the risk of lung cancer than men in the same age range with lower exposures. However, this association was based on a very small population (12 lung cancer cases and 62 controls) and should not be considered definitive results. The data also suggest that women under the age of 65 who had over 10,000 pCi/l of radon in their water supply also have an increased risk of lung cancer, although this was not a statistically significant association, These findings do suggest that radon-induced lung cancers occur at an earlier age (the mean age of the lung cancer cases in the study was 63.3).

There was no significant synergistic effect demonstrated between high radon levels and cigarette smoking. (As expected, lung cancer patients had smoked four times as much as the controls and male patients with other cancers had smoked almost twice as much as controls.)

No significant increase in the risk of other forms of cancer was indicated for persons exposed to high levels of radon in either air or water.

This research is continuing and the data will be reanalyzed when data on 100 lung cancer cases have been collected.

TABLE OF CONTENTS

LIST OF FIGURES

FIGURE PAGE

LIST OF TABLES

ACKNOWLEDGEMENTS

This research was made possible by grants from the Maine State Legislature, Augusta; the Guy Gannett Foundation, Portland; the American Cancer Society, Maine Division, Brunswick; the Maine Cancer Research and Education Foundation, Portland; the Maine Department of Human Services, Augusta; the Maine Lung Association, Augusta; and the Maine Medical Center Cancer Research Fund, Portland.

My special gratitude goes to the coinvestigators of the project and their associates: Dr. C.T. Hess, Professor of Physics, and Steven Lawrence at the University of Maine, Orono; Dr. Peter W. Rand, Director of Research, and Eleanor Lacombe at the Maine Medical Center, Portland; and Dr. Gregory F. Bogdan, Epidemiologist, and Robert Wood, of the Environmental Health Unit of the Maine Department of Human Services.

My gratitude also goes to Walter A. Anderson, State Geologist, and Andrews L. Tolman, Director of the Hydrogeology Division of the Maine Geological Survey for their support of the project, to Dr. Marc C. Loiselle for sharing his expertise in igneous petrology, and to Robert D. Tucker, Director of Cartography, John Poisson and Bennett Wilson for their excellent graphics.

INTRODUCTION

Radon-222, a radioactive gas and naturally-occurring decay product of uranium, has been associated with lung cancer in miners from Czechoslovakia, Canada, Sweden and the United States (22,11,21,14). However, the question remains as to whether the radon levels found in the domestic environment pose a significant health risk. Research conducted in Sweden and Maine, two areas where high levels of radon have been observed in homes, suggest that an excess incidence of lung cancer (19,9) and total cancers (9) is associated with radon, but the results are inconclusive.

The first part of this paper gives background information on radon: what radon is, how it enters the home, and what research has been done on its health effects. The second part describes the epidemiological study conducted in Maine to determine if radon at the levels found in the home increases the risk of lung cancer and of total cancers combined. For this study, radon levels were measured in the air and water in the homes of three groups of patients: lung cancer patients, other cancer patients, and non-cancer patients. The purpose was to determine if the cancer patients had been exposed to significantly higher levels of radon than the control group. Data on potentially confounding variables, including smoking histories and occupational exposures to carcinogens, were collected through questionnaires, and geologic and hydrologic data was examined to improve our ability to predict where high radon levels might occur.

BACKGROUND

PHYSICS AND GEOLOGY

Radon, $_{86}Rn^{222}$, is an inert gas and a radioactive decay product of Uranium-238 (fig.1). Because it is chemically nonreactive, it can migrate from its origin in bedrock and unconsolidated materials and enter the atmosphere via soil gases and ground water. Radon has a half life ($t_{\frac{1}{2}}$) of 3.825 days. When it decays it emits an alpha particle, a helium nucleus, composed of two protons and two neutrons which give it a net charge of +2. The alpha particle given off by radon has an energy of 5.49 MeV. Radon levels are measured in picocuries per liter (pCi/l). (One picocurie of a radioactive element undergoes 2 nuclear disintegrations per minute). The Maine Department of Human Services' suggested standards are 20,000 pCi/l of radon in water supplies and 2 pCi/l in the air in homes.

Two decay products of radon, Polonium-218 ($t_{\frac{1}{2}}$ = 3.05 min.), and Polonium-214 ($t_{\frac{1}{2}} = 1.6 \times 10^{-4}$ sec.), also emit alpha particles with energies of 6.00 MeV, and 7.69 MeV respectively. These two short-lived "radon daughters" deliver the alpha dose believed responsible for the excess bronchogenic lung cancers observed in uranium ore miners. This will be discussed in more detail further on.

In Maine the type of rock most likely to contain concentrations of uranium, and therefore yield potentially dangerous levels of radon, are granites and high-grade metamorphic rocks, particularly those which have undergone partial melting (fig.2).

Most of the granite bodies, or plutons, in Maine were formed 350-400 million years ago from molten magmas rising from the earth's mantle. As the magma started to cool, iron- and magnesium-containing minerals began to crystallize out first. Because of their large size, the uranium atoms did not fit easily into the crystal lattice of these early-crystallizing minerals and so were forced into the still-molten portion of the magma. Silica, aluminum, and certain other light-colored minerals, crystallize later, and it is for this reason that the granites, which contain between 90-100% sialic minerals, may contain high levels of uranium. The last portion of a magma to crystallize contains volatile fluids, which, under high pressure, can force residual magma into fractures and planes of weakness in the country rock surrounding the magma body. Due to these fluids, the magma in the fractures forms coarse-grained pegmatite. In Maine, it is these pegmatite veins associated with granite plutons, that are most likely to contain concentrations of uranium and other rare minerals.

The high-grade metamorphic rocks, which can also contain elevated concentrations of uranium, were created in two ways. First they can form from sedimentary rocks by geologic processes requiring millions of years. Burial of the sediments subjects them to increased temperatures and pressures. If these forces reach a

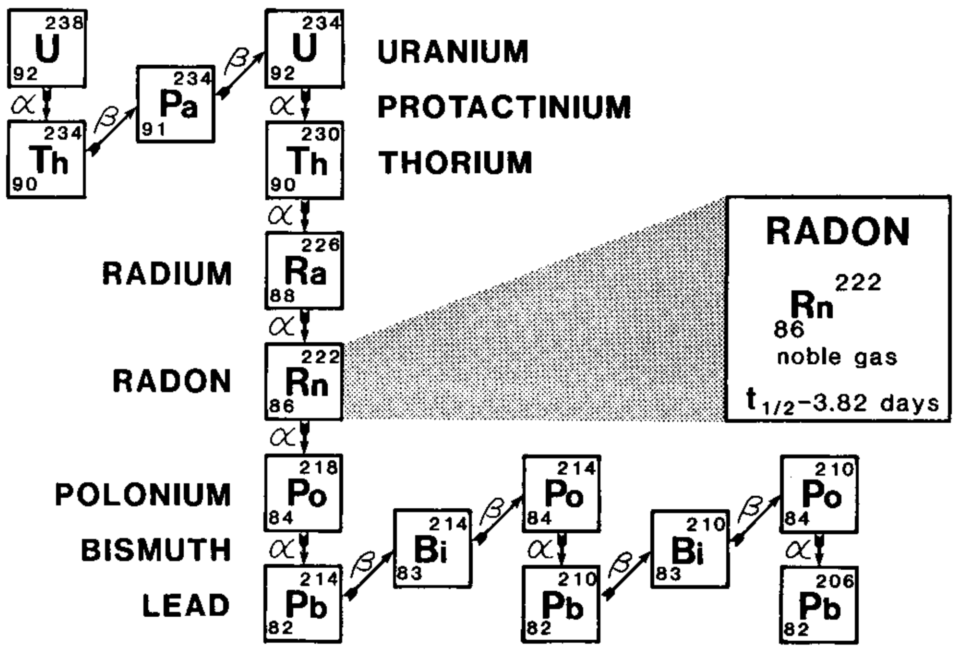

Figure 1. The Uranium Decay Series. Polonium-218 and Polonium-214 are the short-lived radon daughters of concern in the incidence of lung cancer.

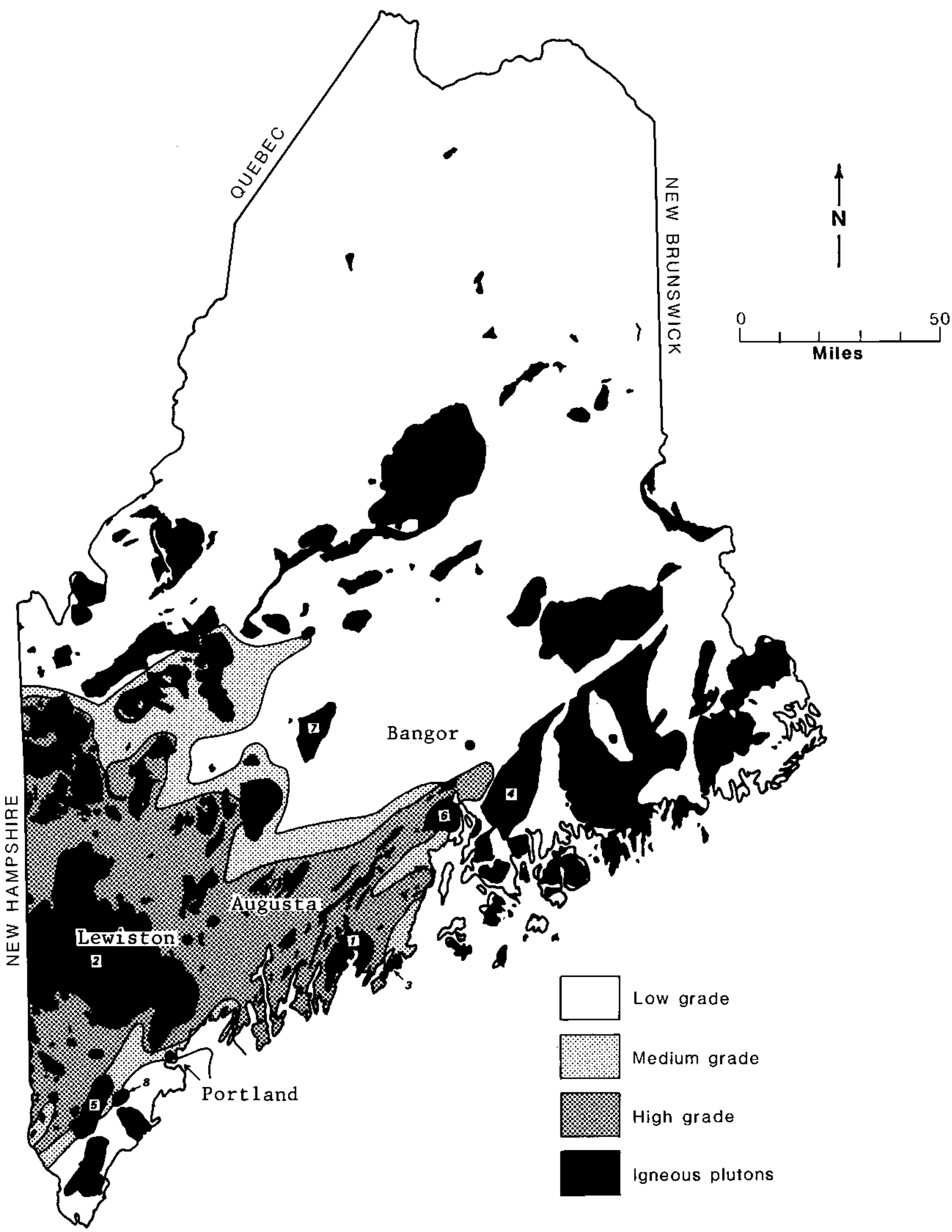

Figure 2. Metamorphic Grade of Bedrock in Maine (from Guidotti (8))

magnitude where the rock once again becomes partially melted, uranium atoms are released from their unstable position in the crystal lattice into the more liquid fraction of the melt, which collects in fractures in the rock. Rock can also become altered by contact with the heat and hydrothermal solutions given off by cooling magmas.

Radon is also present in the overburden, the unconsolidated materials overlying the bedrock. Two factors influencing the amount of radon that reaches the ground surface are the amount of radium at the surface of the soil particles and the permeability of these materials. Research has shown that most soils have the ability to release over 10% of the radon formed, which suggests that much of the radium must be at or near the surface of these particles (17).

Permeability, a measure of interconnected air spaces, increases in proportion to the fraction of coarser materials comprising the overburden. Measurements of uranium tailings have shown that 75% of the radon that migrates to the surface comes from the first 2 meters. It is estimated that in more solid soils containing clay, most of this radon originates in the first meter (17). It has been further demonstrated that emanation is increased with soil moisture levels up to 20% but is retarded at higher levels. Six inches of frozen soil reduces the emanation by 40% (17). The National Council on Radiation Protection and Measurements reports an average emanation rate of 0.42 pCi/m^3/sec. for soils in the U.S. (16).

In Maine, a large portion of the overburden is glacial till, which is composed of varying proportions of clay, silt, sand, and gravel. Studies done in Sweden have recorded high levels of radon in tills composed of uranium-rich granitic materials and in eskers, which are long sinuous ridges of stratified sand and gravel deposited by subglacial rivers (Table I). Near Stockholm, a house built on an esker made up of granite pebbles had 270 pCi/l of radon in the air, which is 25 times the Swedish standard for new homes (2). Maine, too, has a substantial esker system which, along with the coarser tills, may be a significant source of radon.

Table I. Normal Contents of Radon-222 in Swedish Soils, Measured at 1 m Depth (pCi/l)

Till, normal	135 - 811
Till, with granitic material	270 - 1,622
Till, with uranium-rich granitic material	270 - 5,405
Esker gravel	270 - 4,054
Sand, silt	54 - 811
Clay	270 - 2,162
Soils with alum shale	1,351 - 27,027

(from Åkerblom (3))

RADON PATHWAYS INTO THE HOME

There are three principal pathways by which radon can enter the home (fig.3). Potentially the most important source is the overburden discussed above. A team of Swedish researchers concluded that "the radon content of soil gas is always sufficient to give higher radon daughter levels than [10.8 pCi/l, the Swedish standard] <u>if</u> sufficient soil gas leaks into a house, and <u>if</u> the volume of air in the soil is sufficient to maintain active transport" (3). These studies showed that radon in the soil gases does not merely enter the home by passive diffusion as had originally been assumed, but rather it is drawn into the home through cracks and pore spaces in basement walls and floors by the vacuum created by the rising of warmed air. Fans used to vent kitchen fumes magnify the problem by increasing this vacuum. The amount of radon retained in the house is influenced by the volume of the house and the rate of air exchange, the latter being a function of how well the house is insulated. Therefore, ironically, the more fuel-efficient the home, the greater the potential for elevated radon levels.

A second pathway for radon to enter the home is through the domestic water supply, especially if that supply is a well drilled into granite or high-grade metamorphic rock. Under the force of gravity, rainwater infiltrates the unconsolidated materials and works its way to the bedrock where it can accumulate in cracks and fissures. Radon given off at the rock surface can dissolve in this ground water. When these fractures are intercepted by a well, the radon-bearing water is drawn into the house where heating and agitation, such as occur in a dishwasher or shower, release radon into the air. Public water supplies usually have low levels of radon because the source is frequently a lake or other surface water which allows radon to escape readily into the atmosphere. Also, there may be significant retention time in distribution pipes and water tanks, which allows radon and its progeny to decay to low levels. Water from shallow sources (dug wells, springs, and well points) is usually low in radon because of dilution from rainwater and emanation to the atmosphere (Table II).

Table II. Mean Radon Levels in Shallow Wells, Springs, and Public Supplies

SOURCE	MEAN RADON (pCi/l)	N
Dug Wells	1,432	46
Well Points	1,448	5
Springs	3,705	22
Water Companies	1,936	63

(original data from Hess (9))

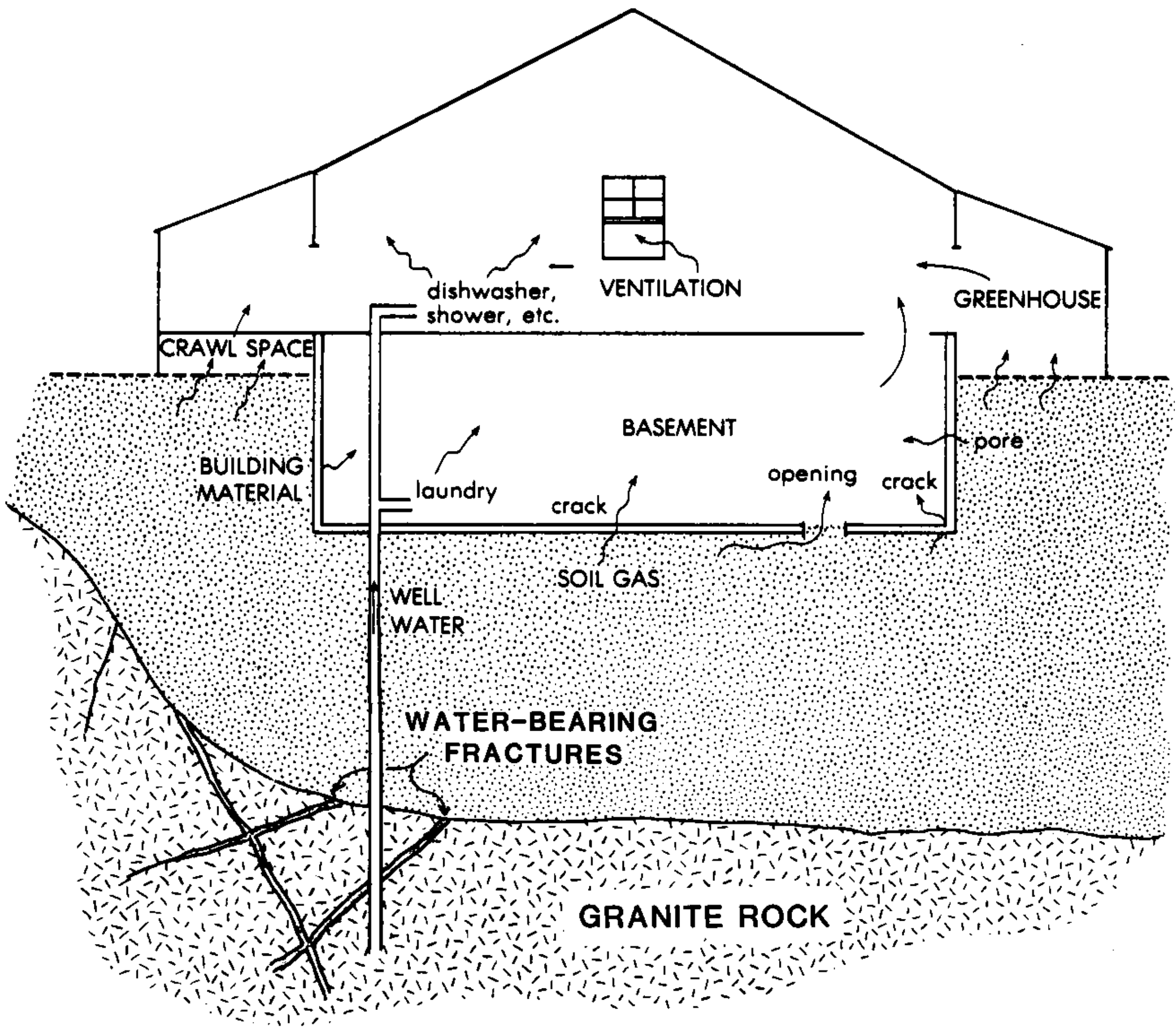

Figure 3. Radon Pathways into the Home (from Weiffenbach (26))

The final and probably least significant source of domestic radon in Maine is building materials. Granite blocks and field stones are commonly used in older homes as foundations, but they have a relatively small surface area from which radon can be released to the air. Few homes are entirely built of these materials. However, in solar-heated homes which use crushed rock as a medium for heat storage, high levels of radon can be given off even from rock containing average amounts of radium (3). Again, this is because of the large surface area and the amount of air circulating around these surfaces. This effect is exacerbated by the fact that solar homes are usually highly insulated to retain heat. One solar home in Maine had a radon concentration of 87 pCi/l, 43 times the limit recommended by Maine's Department of Human Services. These emanations were due primarily to an attached greenhouse with a foundation of gravel over granite bedrock (10).

THE PHYSICAL EFFECTS OF RADON AND ITS PROGENY

In the home, many of the radon daughters plate out on walls and furniture and so never enter the body. Others, however, adhere to smoke, dust, and other particulate matter in the air and are carried into the respiratory tract. The basal cells of the bronchial epithelium are the cells most sensitive to radiation-induced cancers. These cells remain relatively undifferentiated and are capable of becoming mitotically active when there is a necessity for tissue renewal.

Radon itself is soluble in fat and body fluids and has the potential to pass through the alveolar membrane. When it enters the circulatory system it can be transported to other organs of the body, theoretically inducing cancers at these sites (20). Because radon is fat-soluble, breast cancer is potentially another radon-related disease. However, compared to the dose received by the bronchus from radon daughters, the dose received by other organs from radon is very small (Table III).

When radon or its short-lived daughters are deposited on a tissue, the alpha particles emitted cause the closely spaced ionization of water molecules in the cell. If this takes place in the cell nucleus, free radicals then have the potential to damage portions of DNA, which could initiate a malignancy. The types of damage to the DNA include the deletion of a base, the chemical cross-linking of two base pairs, and the breaking of the DNA strand (fig.4). If one strand of the DNA double helix is broken, specialized enzymes, ligases, can usually repair the damage. However, when there is a double strand break, as frequently occurs with alpha radiation, repair may be faulty and the damage irreparable. When radiation damages two adjacent chromosomes or two arms of a chromosome during mitosis, misrepair may separate gene sequences which initiate cell replication from sequences that inhibit this process, permitting an uncontrolled proliferation of

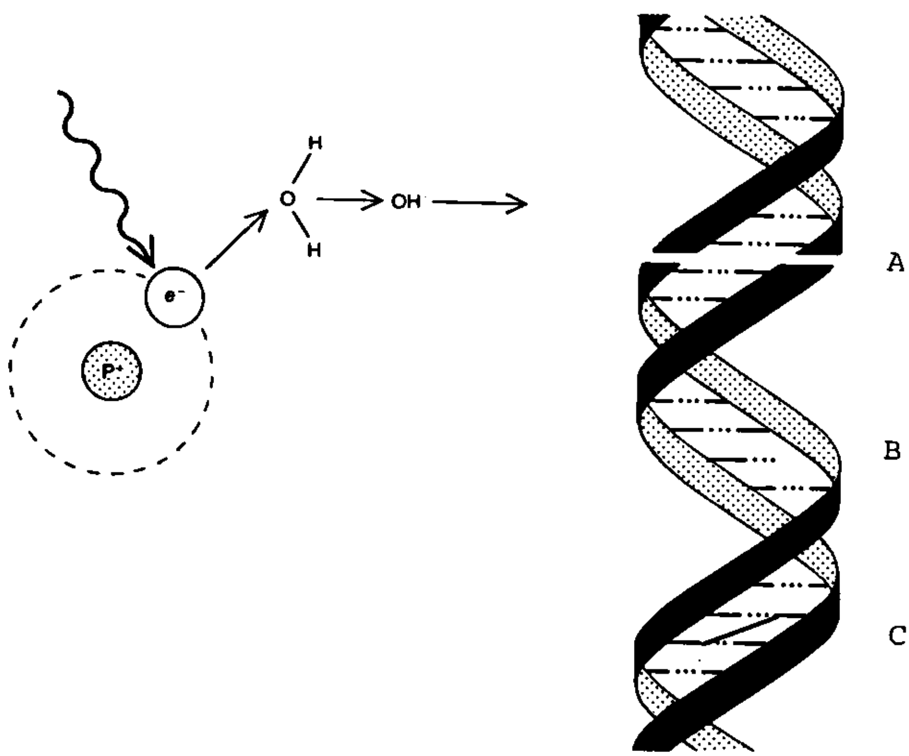

Figure 4. Radiation Damage to DNA. A. A double strand break. B. The deletion of a base. C. The chemical cross-linking of the two DNA strands. (from Upton (25))

Table III. Dose Rate in urad/h from Radon and Radon Daughters to the Bronchioepithelium and Other Organs and Tissues of the Body. The inhalation rate equals 0.23 l/s of air containing 1 pCi/l of radionuclide(s).

ORGAN OR TISSUE	RADON	RN DAUGHTERS
Basal Cells of Bronchioepithelium	---	16
Lungs (Alveolar Tissue)	0.023	3.30
Blood	0.015	0.11
Additional radiation due to passage through the lungs	---	0.33
Liver	0.013	0.090
Kidneys	0.015	0.380
Adrenal Glands	0.034	0.040
Spleen	0.013	0.033
Muscles	0.013	0.013
Bones	0.0034	0.034
Marrow	0.015	0.035
Gonads	0.021	0.008

(from Pohl (20))

cells. Radiation damage might also predispose an individual to a malignancy by inactivating genes responsible for transcribing the ligases (24). Whatever the mechanism, it is age-dependent, as there has been no significant appearance of lung cancer in miners before the age of 40, and the earlier in life a radiation exposure occurs, the longer the latency period before a tumor appears (17).

THE INCIDENCE OF RADON AND RISK ASSESSMENT

The potential for radon-daughter-induced lung cancer is well founded by studies of miners of uranium and other ores. However, the magnitude of this threat in the home environment is more difficult to determine and is usually estimated by extrapolating from occupational exposures and from exposures received by atomic bomb victims.

As early as 1579, a lung disease now identified as bronchogenic cancer was observed among miners of uranium-bearing ores in the Schneeburg and Joachimsthal Valley in Central Europe (1). In the United States, Lundin (14) studied miners of the Colorado Plateau and observed a respiratory cancer mortality rate 6 times higher than that observed in the general public. In northern Sweden, the rate was 14 times higher among iron ore miners whose exposure to radon daughters was 90-180 pCi/l (21). (The International Commission on Radiological Protection recommends a maximum radon level of 30 pCi/l for air in underground mines).

The existence of radon in domestic water supplies has been recognized since the beginning of the century. In England, J.J. Thompson measured radon levels in water wells in 1902 (23), as did H.A. Bumstead and L.P. Wheeler in Connecticut in 1904 (5). Radon was first discovered in Maine's ground water in 1957 when a 13 year-old boy, using a beta-gamma probe, noticed a high count near a pressure tank in Raymond, a town located on a large body of granite, the Sebago Pluton.

Since then, several studies have revealed some exceptionally high radon concentrations in domestic water supplies (7,13,9). One well in Georgetown, an area cross-cut by pegmatite veins, contained a radon concentration of approximately 700,000 pCi/l, 35 times the state's recommended standard.

Preliminary studies on the health effects of radon were done by Hess (9) and by the Maine Department of Human Services (12). Both found a statistically significant correlation between average radon levels and mortality rates from various forms of cancer. Hess correlated age-adjusted respiratory cancer rates with estimated water radon levels for each county and found that as many as 42% of lower respiratory cancers in women may be radon-related. He also found a significant correlation between radon activity and all forms of cancer in women (Table IV and figs. 5,6). His tentative explanation for the higher percentage of radon-related lung cancers among women is that more of men's lung cancers are caused by cigarette smoking, and that women are more often involved in water-related tasks in the home.

Table IV. Correlation Coefficients for Radon and Total Cancer and Radon and Lung Cancer in Maine

CANCER TYPE	N	R	VARIANCE	PROBABILITY
All Cancers:				
Male	17,793	0.47	0.22	> .05
Female	16,135	0.61	0.37	< .05 *
Average	33,928	0.58	0.34	< .05 *
Trachea, Bronchus, Lung:				
Male	3,682	0.46	0.21	> .05
Female	656	0.65	0.42	< .01 *
Average	4,338	0.56	0.31	< .05 *

* statistically significant

(from Hess (9))

A follow-up study done by the state's Department of Human Services using cancer mortality rates for 50 low-radon and 50 high-radon commmunities also showed a significantly high correlation

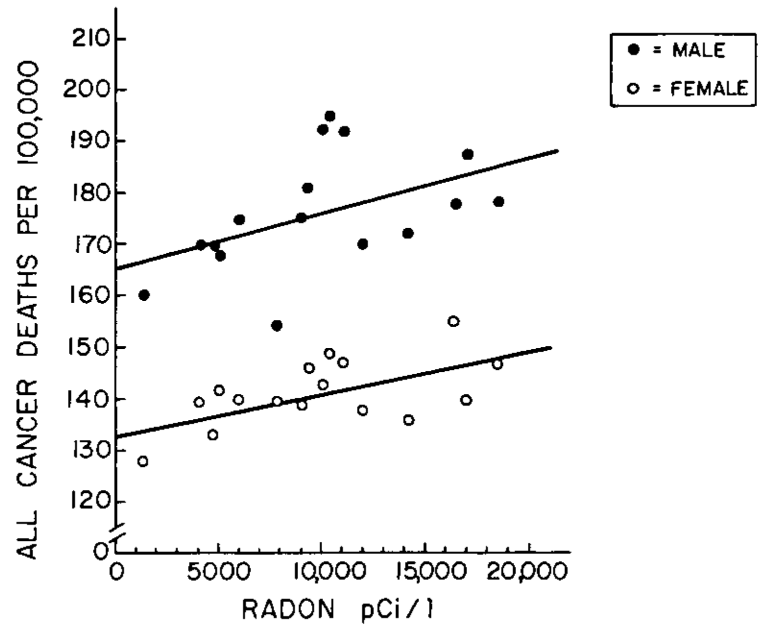

Figure 5. The Relationship of Radon in Water to All Cancer Deaths (from Hess (9))

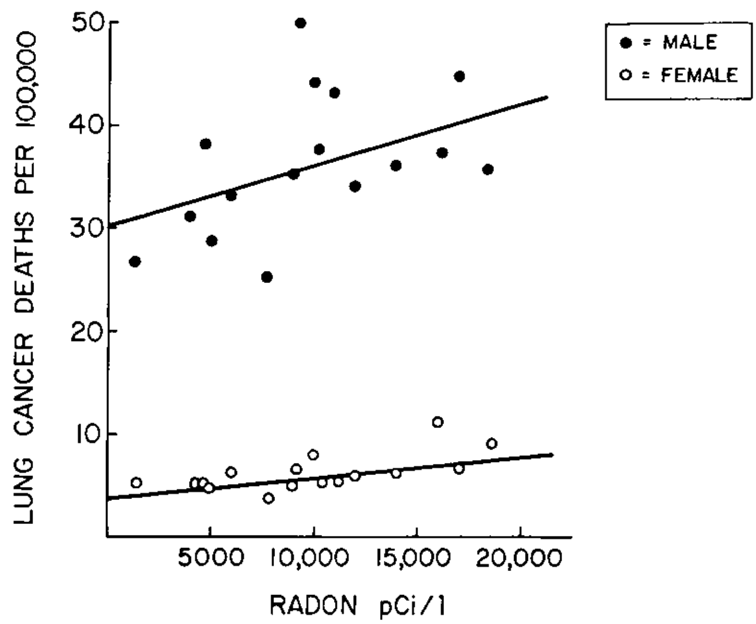

Figure 6. The Relationship of Radon in Water to Lung Cancer Deaths (from Hess (9))

between radon in water and total cancers in women, as well as lymphatic and hematopoietic cancers in females, genito-urinary cancers in men and all other cancers in men.

Although correlations using regional averages suggest that domestic radon is a cause of various cancers, and epidemiological studies involving miners implicate radon daughters in the etiology of lung cancer, there have been very few epidemiological studies undertaken to determine directly if individual cancer patients have been exposed to elevated levels of radon in their homes. A study done by Axelson (4) in Sweden revealed that a rural population living in stone dwellings with basements, which are often used as living areas, experienced a lung cancer mortality rate 5 times higher than that for persons living in wooden dwellings without basements. A second Swedish study (19) looked at two groups of cases and matched controls, and compared their estimated radon exposure based on house construction. The first group consisted of 53 pairs of identical twins, one of which had died of lung cancer. No differences in radon exposure could be determined between cases and controls for either smokers or non-smokers. However, the use of identical twins may have introduced a selection bias, because both twins spent the early part of their lives in the same dwelling. A second group of 30 lung cancer patients were matched to controls with whom they had no blood relationship. No difference in exposure could be determined between the non-smoking cases and controls, although the cases who smoked had a significantly higher estimated radon exposure than the controls matched for similar smoking histories. This suggests that radon may act as a co-carcinogen to produce a malignancy.

A third Swedish study (6) directly measured the radon levels in the homes of 23 lung cancer patients and 202 controls which had died of other causes. It was found that in houses with radon daughter concentrations equal to or exceeding 1.35 pCi/l, the risk of developing lung cancer was 2.6 times higher for non-smokers than for the general population, and 10.3 times higher for smokers. They concluded that "the major role of tobacco smoke could be that of a promoter, whereas radiation acts as the major cancer initiating agent."

As in this latter research, Maine's epidemiological study has the advantage of actual radon measurements taken in each participant's home, rather than relying on estimates from generalized data. A description of this study follows.

THE EPIDEMIOLOGICAL STUDY

METHODS

DATA COLLECTION: Initially, potential participants were made aware of this study by the distribution of cards explaining the purpose of the research to the admissions offices of the Maine Medical Center, the Southern Maine Radiation Institute, and private oncologists' offices in the Portland area. Later in the study, patients were also solicited from the Eastern Maine Medical Center in Bangor, Central Maine Medical Center in Lewiston, and other regional hospitals, in order to include enough lung cancer patients for a meaningful statistical comparison. To be eligible for inclusion in the study, patients had to have been over 35 years old and on a privately owned drilled well for at least 10 years. Aroostook County residents were excluded from the study because previous studies indicated that water radon levels there were exceptionally low. After qualified persons were located, the Maine Medical Center contacted the patient's physician to confirm the diagnosis stated on the returned card, to acquire the results of tests performed on the patient, and to record the histology of the tumor if the patient had lung cancer. Subject to the approval of the physician, the patient was then administered a questionnaire. Originally this was done by a trained interviewer, but later, to save time and expense, the questionnaires were mailed to the patient's home, and a follow-up call was made if they were returned with blanks or ambiguous answers. The questionnaire requested basic demographic data, information on the house and water well construction, medical history, smoking habits, and occupational exposure to carcinogenic agents (see Appendix).

After a map locating the patient's home was received with the questionnaire, air and water samples were collected by a graduate student from the University of Maine at Orono. Radon levels in duplicate water samples were measured at the University using the liquid scintillation method. A track-etch cup was used to measure the air radon and was placed in the room which receives the most use and which is most apt to contain high levels of radon - usually the kitchen. These cups have a strip of sensitized plastic on the bottom, which becomes exposed on contact with alpha radiation. Each cup is left in place for approximately 2 months, after which time they are sent back to Orono and from there sent in batches of 50 to the manufacturer, the Terradex Corporation. There the degree of exposure is enhanced by chemical etching, and the radon levels are calculated from the number of pits thus produced in the sensitized plastic.

The final step was to collect geologic data for each well location. This included the age, formation, lithology, and most importantly, the metamorphic grade of the bedrock, as well as the type, glacial form, and thickness of the overburden. These data were taken from detailed bedrock and surficial geologic maps compiled through the Maine Geological Survey. The well depth, well yield, and casing length (an estimate of the overburden thickness) was requested in the patients' questionnaires.

DATA ANALYSIS: Geology and Hydrology. To determine where water radon levels are most likely to be high, the mean, median, maximum and minimum water radon levels were calculated for each classification of the geologic age of the bedrock, of its lithology (such as limestone, sandstone, etc.), of its metamorphic grade, and for the igneous pluton nearest the well. Descriptive statistics were also calculated for ranges of depth and yield of all the wells, as well as the type and thickness of the overburden. A second set of statistics was calculated just for wells drilled into igneous rocks. A regression was run to determine the relative importance of ground water as a source of airborne radon, and the average air radon levels were calculated for each major overburden type as an indication of the radon contribution from the soils.

Epidemiology. The first analysis was on the raw data to determine if either group of cancer patients was exposed to higher levels of airborne radon than the controls. Histograms were constructed to show the distribution of air radon values among the three groups. Then, because the distributions were found to be skewed to the right and were of samples of unequal size, the significance of the difference in distribution was determined using the Kruskal-Wallis test for ranked data instead of a standard ANOVA.

The next step in the analysis was to determine what, if any, confounding variables might have influenced or masked a relationship between radon exposure and the incidence of cancer. The most obvious factor was tobacco smoking. Others were: occupation, age, sex, educational level as an estimate of social class, and length of time at the residence.

The means and standard deviations of each of these variables were calculated for each patient group and those variables that were significantly different among groups, based on a standard ANOVA, were stratified (divided into subgroups). For each of these subgroups, 2X2 frequency tables were constucted (one for air radon and one for water radon) with lung cancer and other cancer cases compared to controls for their exposure to high and low radon values. High radon was considered to be >3 pCi/l in air (based on the U.S. Environmental Protection Agency's suggested standard) and >10,000 pCi/l in water (based on research by Hess). Odds ratios were calculated as illustrated by the generalized diagram below:

	CASE	CONTROL	
HIGH RADON	a	b	m1
LOW RADON	c	d	m2
	n1	n2	n

Odds Ratio = ad/cb

An odds ratio was considered statistically significant if the chi-square value exceeded 3.84 ($p < .05$):

$$X^2 = ((ad-cb)\ n)^2/(n1\ n2\ m1\ m2)$$

RESULTS

GEOLOGY AND HYDROLOGY. Little variation in water radon levels could be seen among the various geologic ages of bedrock. However, there was a tripling in the mean water radon value from low grade to high grade metamorphic rocks and a doubling from high grade metamorphic rock to igneous rock (Table V). Figure 7 also illustrates the increasing frequency of high radon water from wells in increasingly higher metamorphic grade rock. Among the various igneous plutons there was considerable variation, with the wells on or near the Waldoboro Pluton having both the highest mean and median water radon values (Table VI). The next highest average radon was in water from wells on or near the Sebago Pluton. There was also considerable variation within plutons.

Table V. Mean and Median Water Radon Values by Metamorphic Grade of Bedrock (pCi/l)

GRADE	N	MEAN	SD	MEDIAN
Low	95	2,111	2,211	1,682
Medium	76	4,093	6,144	2,560
High	102	7,059	11,096	3,192
Igneous	114	15,008	22,577	6,546

Table VI. Mean and Median Water Radon Levels by Pluton (pCi/l)

PLUTON	N	MEAN	SD	MEDIAN
1: Waldoboro	24	18,642	18,659	13,468
2: Sebago	98	13,323	22,566	4,860
3: Spruce Head	9	10,060	18,423	3,637
4: Lucerne	15	6,820	16,710	1,357
5: Lyman	23	5,778	10,115	2,085
6: Mount Waldo	5	4,035	4,796	2,705
7: Hartland	12	1,540	1,162	1,062
8: Saco	5	1,529	606	1,632

(pluton numbers refer to locations in figure 2, p.4)

There was little association between water radon levels and well and overburden characteristics either for the total wells combined (Table VII) or for wells drilled into granitic rock (Table VIII), although both the mean and median radon levels in granite wells tended to be lower when the overburden was sand and gravel

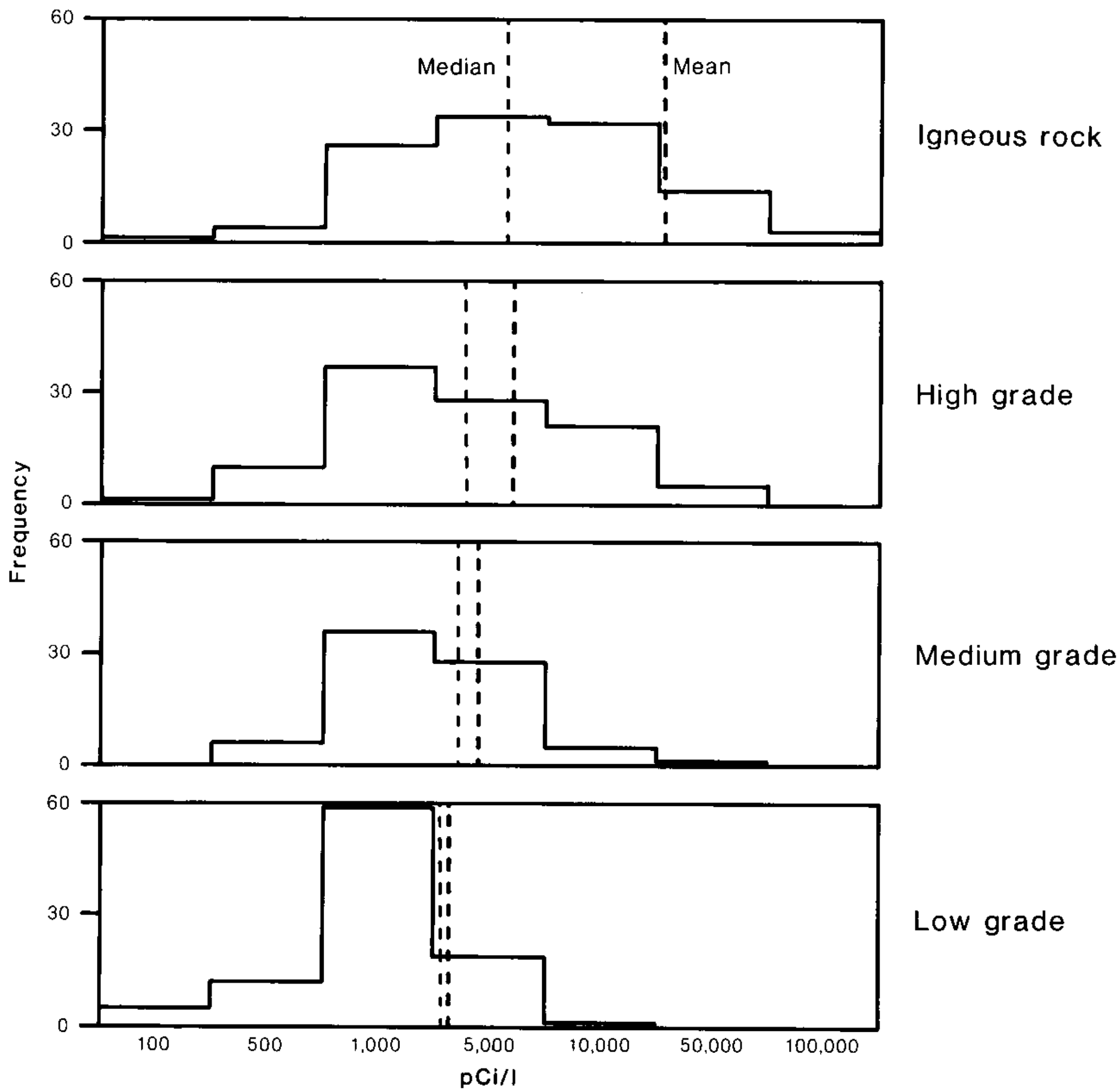

Figure 7. Frequency Distribution of Radon Concentrations in Water from Wells in Low-, Medium-, and High-grade Metamorphic Rock and Igneous Rock (apparent distortion in median due to non-linear scale)

Table VII. Radon Concentration in All Wells by Well and Overburden Characteristics

	Number of Samples	Radon Concentration (pCi/l)			
		Mean	Median	Minimum	Maximum
Well Depth (feet)					
<100	160	6,826	2,500	107	65,656
100-200	142	8,685	3,031	190	151,182
200-300	56	5,148	2,712	384	40,069
300-400	16	13,846	5,548	418	77,902
>400	8	10,363	2,196	142	32,675
Well Yield (Gallons Per Minute)					
<2	159	8,330	3,090	107	77,902
2-5	54	7,348	2,512	478	121,522
5-10	22	8,619	1,485	462	51,638
10-25	31	8,883	3,444	527	65,280
25-50	21	5,735	2,550	372	38,761
>50	9	2,358	2,016	190	7,801
Unknown	86	6,859	3,055	192	151,182
Overburden Type					
Till	229	8,931	3,344	107	121,522
Clay	79	6,119	2,119	192	151,182
Sand and Gravel	46	4,892	2,163	190	36,782
Other or missing	28	5,878	2,451	480	77,902
Overburden Thickness (feet)					
0-10	261	7,833	2,546	107	121,522
10-20	57	9,310	3,304	351	151,182
20-50	35	5,084	3,040	192	32,036
50-100	23	6,396	3,071	190	36,782
>100	5	2,322	1,858	1,147	5,023

Table VIII. Radon Concentration in Igneous Rock Wells by Well and Overburden Characteristics

	Number of Samples	Radon Concentration (pCi/l)			
		Mean	Median	Minimum	Maximum
Well Depth (feet)					
<100	43	12,595	7,015	543	65,280
100-200	42	19,219	6,997	341	151,182
200-300	15	7,306	3,750	614	38,761
300-400	9	21,789	16,054	743	77,902
>400	4	13,312	10,215	142	32,675
Well Yield (Gallons Per Minute)					
<2	48	15,555	7,398	142	77,902
2-5	13	16,817	7,015	1,502	121,522
5-10	6	21,605	16,322	1,119	51,638
10-25	11	16,435	6,533	743	65,280
25-50	6	12,705	3,161	1,047	38,761
>50	2	3,859	-	2,016	5,702
Unknown	27	12,894	5,023	1,348	151,182
Overburden Type					
Till	72	16,634	8,158	142	121,522
Clay	18	15,938	4,588	614	151,182
Sand and Gravel	17	7,099	5,023	1,528	32,675
Other or missing	6	17,087	4,550	909	77,902
Overburden Thickness (feet)					
0-10	77	15,720	6,968	142	121,522
10-20	14	24,189	10,022	1,348	151,182
20-50	11	8,704	3,533	1,308	32,036
50-100	8	6,178	4,586	1,163	17,179
>100	2	3,440	-	1,858	5,023

than when the overburden was till. This could be explained by the fact that sand and gravel deposits are more permeable and allow radon to migrate faster to the surface.

However, water supplies do not appear to be the primary source of airborne radon. A Model I Regression was run for air and water radon, yielding the following equation:

$$Y = 1.79 + 6.56 \times 10^{-5} X \qquad (r^2 = 0.156)$$

Y = air radon
X = water radon

These results indicate that approximately 16% of the variance in air radon levels is attributable to the variation in the water radon levels, and that radon in the air is increased by 1 pCi/l for every 15,000 pCi/l of radon in water. Although this model is not strictly appropriate for these data, it does suggest that other sources yield significant amounts of radon to the air in homes.

Additional data suggests that the soil may be more important than water as a source of air radon. Table IX shows the mean, median, and 75th percentile for air radon values in homes built over sand and gravel, till, and clay. All the parameters for air radon increase in value as the soil materials increase in permeability.

Table IX. Mean, Median, and 75th Percentile Values of Airborne Radon by Overburden Type (pCi/l)

TYPE	N	MEAN	MEDIAN	75%
Sand, Gravel	46	3.03	1.83	4.41
Till	230	2.31	1.64	2.72
Clay	82	1.82	1.39	2.09

$p < .05$

EPIDEMIOLOGY. Between June 1982 and January 1985, 343 patients who met the original selection criteria had joined the study: 36 lung cancer patients, 124 other cancer patients and 183 non-cancer patients. Of the "lung" cancer patients, 35 had cancer of the trachea, bronchus or lung, and one had cancer of the pleura. Of the other cancer patients, 14% (17) had colonic or rectal cancer, 19% (23) had breast cancer, 28% (35) had cancer of the genito-urinary system, and 13% (16) had lymphatic or hematopoietic cancer. The remaining 26% were divided among other disease categories, each of which included less than 5% of the patients.

Among the non-cancer patients, 24% (44) had diseases of the circulatory system, including atherosclerotic heart disease (9%), 18% (33) had diseases of the musculo-skeletal system, including osteoarthritis (10%), 14% (25) had diseases of the genito-urinary system, 9% (16) had diseases of the digestive system, and 7% (12)

were free of disease. Each of the other disease categories, including benign tumors (4%), had less than 5% of the patients.

Histograms of air and water radon levels for each patient group showed that the distribution in each case was skewed to the right (fig.8), so the median values were included with the descriptive statistics. When the three groups were compared, there were no statistical differences among the groups in radon levels in air or water, although the high median air value for male lung cancer patients approaches statistical significance (p=0.07) (Table X).

Table X. Mean and Median Air and Water Radon Values by Patient Group and Sex (pCi/l)

		FEMALES			
		AIR		WATER	
GROUP	N	MEAN	MEDIAN	MEAN	MEDIAN
LUNG CANCER	12	1.80	1.36	6,826	5,064
OTHER CANCER	64	2.48	1.82	6,010	2,548
NON-CANCER	85	2.32	1.60	6,332	2,443
		p=ns	p=ns	p=ns	p=ns

		MALES			
		AIR		WATER	
GROUP	N	MEAN	MEDIAN	MEAN	MEDIAN
LUNG CANCER	23	2.26	2.13	6,096	3,689
OTHER CANCER	54	2.10	1.34	9,306	3,001
NON-CANCER	89	2.61	1.78	11,111	3,859
		p=ns	p=ns	p=ns	p=ns

To determine if confounding variables could be masking the effect of radon on the incidence of cancer, the mean packyears (YEARS SMOKED X # CIGARETTES / DAY / 20) for each group were compared, as well as the percent of men and women in each group, the mean age, years at present residence, years of education, and the type of occupation (Table XI). Occupations were grouped by their 1980 Standard Occupational Classification Code as "White Collar" (003-389), "Blue Collar" (403-889) and "Housewives" (914).

SMOKING HISTORY. As would be expected, the lung cancer patients and the other cancer patients were more frequently smokers than the controls. Ninety-two percent (92%) of the female lung cancer patients and 96% of the men smoked, as opposed to 49% and 88% of women and men, respectively, in the other cancer group; 47% of the female controls and 76% of the male controls were smokers. When packyears were calculated, it was found that both lung cancer

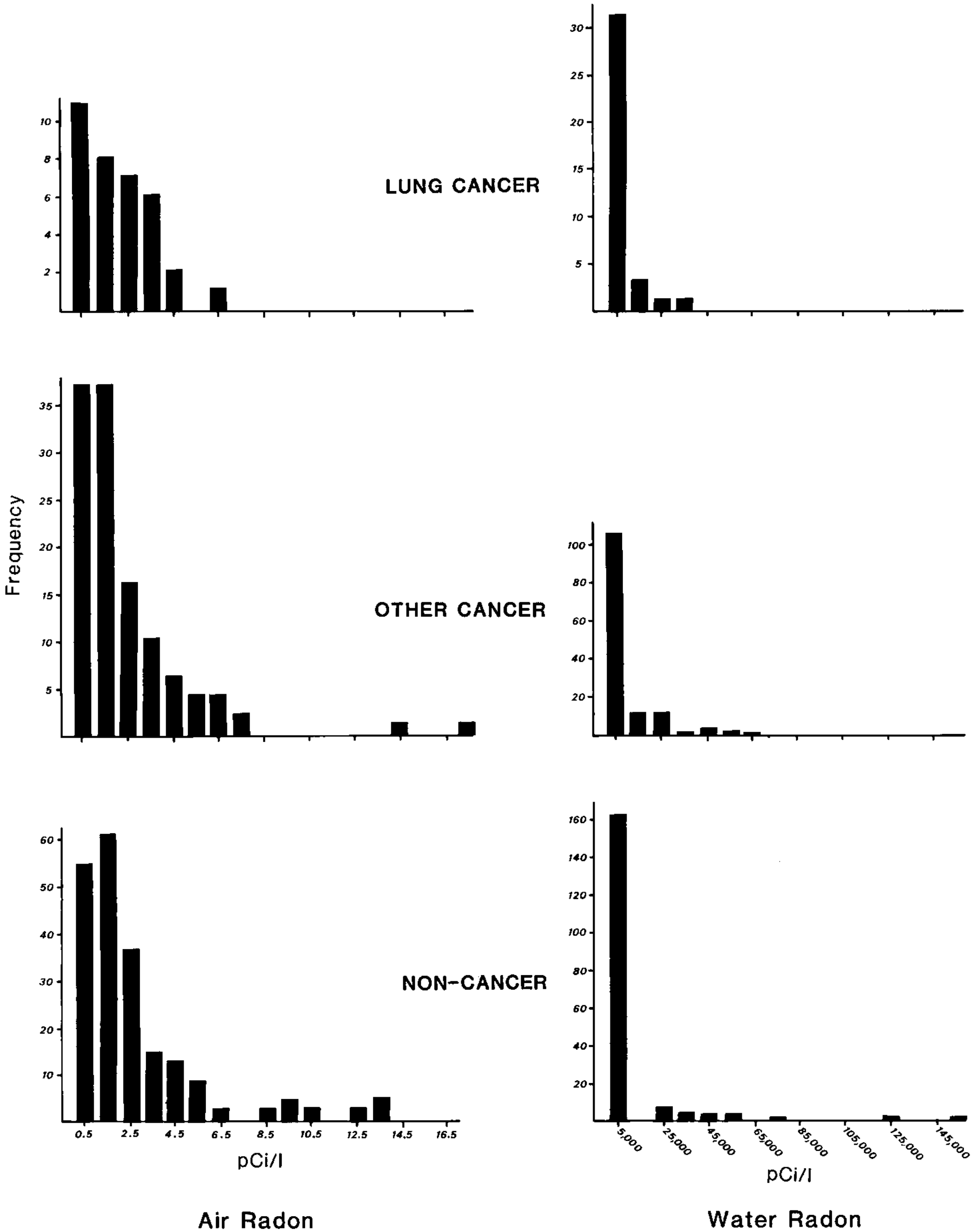

Figure 8. Distribution of Air and Water Radon Values Among Patient Groups

Table XI. Significance of Potentially Confounding Variables

FEMALES	LUNG CANCER	OTHER CANCER	NON-CANCER	SIGNIFICANCE
(N, % of Group)	13 (36%)	68 (55%)	87 (48%)	
Mean Age	63.5	59.3	55.4	NS
Mean Years at Present Residence	16.7	22.8	19.7	NS
Mean Years of Education	12.5	12.6	13.2	NS
Mean Packyears	24.8	7.9	5.6	< .001
Occupation				
White Collar	30.8	37.3	50.6	
Blue Collar	15.4	29.8	18.4	
Housewife	53.8	29.9	28.7	< .005

MALES	LUNG CANCER	OTHER CANCER	NON-CANCER	SIGNIFICANCE
(N, % of Group)	23 (64%)	56 (45%)	96 (52%)	
Mean Age	63.7	66.6	58.9	< .001
Mean Years at Present Residence	20.9	21.3	20.4	NS
Mean Years of Education	11.3	12.5	13.9	< .001
Mean Packyears	50.0	23.9	13.5	< .001
Occupation				
White Collar	43.5	46.5	50.0	
Blue Collar	56.5	49.9	49.0	NS

patients and other cancer patients smoked more heavily than the controls. The means for the three groups were 40.9, 15.1 and 9.8 packyears respectively. In each patient category women smoked less heavily than did the men.

SEX. There was a sexual bias within the lung cancer group but not within the other cancer or control group. Sixty-four percent (64%) of the lung cancer cases were men, while 45% of the other cancer cases and 52% of the controls were men.

AGE. Age also proved to be a confounding variable. The mean age for patients in both the lung cancer and other cancer groups was approximately 6 years more than for the controls (63.3, 62.6 and 57.3 years respectively). When the groups were further broken down by sex, the females of the other cancer group were approximately 8 years younger than their male counterparts. The age difference among men was highly significant.

LENGTH OF TIME AT PRESENT RESIDENCE. The difference in residence time among the groups was not significant.

EDUCATION. The women's level of education among the three groups was not significantly different: 12.5 and 12.6 years for the two cancer groups, and 13.2 for the controls. However, the difference was highly significant for the males. The most highly educated of the subgroups were the male controls, with an average of 13.9 years (1.9 years of college). The male lung cancer group had the least education, an average of 11.3 years.

OCCUPATIONS. The difference in occupation was tested for significance by a chi-square analysis. No significant difference in general occupational category was observed among the men, but the difference was highly significant among women: housewives comprised 54% of the female lung cancer group, 30% of the other cancer group, and only 9% of the female control group.

To compensate for the differences in these variables among the groups, the data were stratified as follows (Table XII):
1) by sex and age (<65 or >65), 2) by smoking level (Non-smoking, Light = 1-14 packyears, Moderate = 15-30 packyears, Heavy = 30+ packyears), and 3) by occupation as previously defined.

Table XII. Odds Ratios for Stratified Data

BY AGE AND SEX

		RADON	LC	NC	ODDS RATIO	CHI-SQ	95% CI
		HI	2	7			
	F <65	LO	4	51	3.643	2.035	0.56-23.69
		HI	3	14			
	M <65	LO	9	48	1.143	0.033	0.27-4.80
WATER							
		HI	0.5	3.5			
	F >65	LO	7.5	26.5	0.505	0.197	0.02-10.90
		HI	0.5	9.5			
	M >65	LO	11.5	25.5	0.117	2.816	0.01-2.18
		HI	1	15			
	F <65	LO	5	43	0.573	0.245	0.06-5.31
		HI	7	9			
	M <65	LO	5	53	8.244	11.391*	2.14-31.73
AIR							
		HI	1	6			
	F >65	LO	6	23	0.639	0.148	0.06-6.37
		HI	0.5	8.5			
	M >65	LO	11.5	26.5	0.136	2.336	0.01-2.55

Table XII. Odds Ratios for Stratified Data (cont.)

BY SMOKING LEVEL

		RADON	LC	NC	ODDS RATIO	CHI-SQ	95% CI
		HI	3	17			
	NON	LO	8	95	2.096	1.076	0.50-8.70
		HI	1	4			
	LIGHT	LO	1	16	4.000	0.932	0.20-78.80
WATER		HI	0.5	6.5			
	MOD	LO	3.5	19.5	0.429	0.303	0.02-9.45
		HI	1	5			
	HEAV	LO	19	18	0.189	2.497	0.02-1.78
		HI	4	21			
	NON	LO	7	91	2.476	1.919	0.66-9.24
		HI	0.5	8.5			
	LIGHT	LO	2.5	12.5	0.294	0.635	0.01-6.92
AIR		HI	1	6			
	MOD	LO	2	19	1.583	0.124	0.12-20.69
		HI	4	2			
	HEAV	LO	16	21	2.625	1.139	0.43-16.16

BY OCCUPATION

		RADON	LC	NC	ODDS RATIO	CHI-SQ	95% CI
		HI	2	14			
	WHITE COL	LO	11	58	0.753	0.119	0.15-3.79
		HI	1	12			
WATER	BLUE COL	LO	12	43	0.299	1.137	0.04-2.53
		HI	1	3			
	HOUSEWIFE	LO	6	23	1.278	0.039	0.11-14.59
		HI	4	15			
	WHITE COL	LO	9	57	1.689	0.626	0.46-6.25
		HI	4	12			
AIR	BLUE COL	LO	9	43	1.593	0.468	0.42-6.08
		HI	1	4			
	HOUSEWIFE	LO	6	22	0.917	0.005	0.09-9.81

The odds ratio was significantly high (8.2) for men under 65 who were exposed to over 3 pCi/l of airborne radon in their homes. To be sure this effect was not due to a spurious association with cigarette smoking, the data were further stratified by smoking level. The results showed that men under the age of 65, whose smoking level was less than 25 packyears, and who were exposed to high levels of radon, still had a relative risk of 8.063 ($X^2 = 5.665$). This suggests that cigarette smoking was not a confounding variable and that the increased risk was due to radon exposure. The odds ratio was high (3.6) but not significant for women under 65 who were exposed to high levels of radon from water, and was low but not significant for men 65 or older who had been exposed to high levels of radon in water or air.

Although the odds ratios increased as smoking levels increased from light to heavy, the relative risk for non-smokers who were exposed to high levels of airborne radon was approximately the same as that for heavy smokers. None of the odds ratios for data stratified by smoking levels were statistically significant.

CONCLUSIONS

The best geologic predictor of where water radon levels are likely to be high, is the metamorphic grade of the bedrock. Radon levels triple from low- to high-grade metamorphic rock and double from high-grade metamorphic to igneous rock, specifically granites. This is a function of the degree of melting during the formation of the rock, which concentrates uranium. Other geologic and hydrologic parameters showed little association with water radon levels.

However, the radon in water supplies contributed only 16% of the radon found in the air (1 pCi/l in the air for every 15,000 pCi/l in the water). The outgassing of the soil appeared to be a more significant source of radon in the home, with air radon levels increasing with the permeability of the overburden on which the home was built.

Despite the small size of the population studied, a statistically significant association was found between high radon levels and the incidence of lung cancer. Men under the age of 65 who were exposed to over 3 pCi/l of airborne radon in their homes had 8.2 times the risk of lung cancer than men exposed to lower levels. This association remained even after the data were further stratified to control for cigarette smoking. Although it was not statistically significant, the data suggests that women under the age of 65 who are exposed to water with radon levels exceeding 10,000 pCi/l had a risk of lung cancer several times higher than that for women exposed to lower levels. This could be due to the fact that more women are involved in water-related tasks in the home than are men, as suggested by Hess (9). This is also consistent with the fact that approximately twice as many female lung cancer patients were housewives than were the female controls, although stratifying for occupation revealed no increased risk. These relative risks suggest that radon-daughter-induced damage to the lung lowers the age at which lung cancer is likely to occur.

There did not appear to be a strong synergistic effect between cigarette smoking and exposure to high radon levels. The odds ratios comparing high and low air radon exposures did increase as cigarette smoking levels increased from light to moderate to heavy, but the relative risk for non-smokers was approximately the same as that for heavy smokers. None of the odds ratios resulting from data stratified by smoking levels were statistically significant.

The observation that over 90% of the lung cancer patients in this study had been cigarette smokers, and that, on the average, they smoked four times as much as the controls, further substantiates the well established fact that cigarette smoking is the primary cause of lung cancer. Males in the other cancer group smoked twice as many packyears as the male controls, which suggests that cigarette smoking contributes to other forms of cancer as well.

This study will be continuing until information on one hundred lung cancer cases has been collected. The data will be reanalyzed at that time.

Future epidemiological studies must be of a larger population to allow more detailed stratification. Ideally, this population would consist of non-smokers, and controls should be randomly chosen instead of self-selected. Measurements of radon in the soils should be included with air and water tests.

REFERENCES

1. Agricola, G., 1597: De Re Metallica. Cited by Heuper, 1942, and as translated by H.C. Hoover and L.H. Hoover, Dover Publication, N.Y., 1950.

2. Åkerblom, G., Aug.24, 1984, personal communication.

3. Åkerblom, G., P. Andersson, and B. Clavensjo, 1983: Soil Gas Radon - a Source for Indoor Radon Daughters. Geological Survey of Sweden, Uppsala, p.1.

4. Axelson, O., C. Edling, H. Kling, L. Andersson, and A. Ringner, 1981: Lung cancer and radon in dwellings. Lancet, II:995-996.

5. Bumstead, H.A. and L.P. Wheeler, 1904: Amer. Journal Science, 17:97.

6. Edling, C., G. Wingren and O. Axelson, 1984: Radon daughter exposure in dwellings and lung cancer. In: Proceedings of the Third International Conference on Indoor Air Quality and Climate, Vol. 2. Stockholm, Sweden, pp. 29-33.

7. Grune, W.N., F.B. Higgins and B.M. Smith, 1960: Natural Radioactivity in Groundwater Supplies in Maine and New Hampshire. U.S. Public Health Service, Contract No. 73551.

8. Guidotti, C.V., 1985: Generalized map of regional metamorphic zones. In: Bedrock Geologic Map of Maine, Osberg, P.H., A.M. Hussey II and G.M. Boone, eds. Maine Geological Survey, Augusta, Maine.

9. Hess, C.T., S.A. Norton, W.F. Brutsaert, R.E. Casparius, E.G. Coombs and A.L. Hess, 1979: Radon-222 in Potable Water Supplies in Maine: The Geology, Hydrology, Physics and Health Effects. Land and Water Resources Center, University of Maine, Orono, pp. 90-98.

10. Hess, C.T., S.A. Norton, W.F. Brutsaert, J.E. Lowry and C.F. Weiffenbach, 1981: Investigations of Radon-222, Radium-226, and Uranium in Air and Water in Maine. Land and Water Resources Center, University of Maine, Orono, 33 pp.

11. Hewitt, D., 1976: Radiogenic lung cancer in Ontario uranium miners 1955-1974. In: Report of the Royal Commission on the Health and Safety of Workers in Mines. Ham, J.A., ed. Ministry of the Attorney General, Province of Ontario, Toronto, Canada, p. 78, 319.

12. Hinckley, W.W., 1982: Does Radon in Drinking Water Cause Cancer? Maine Bureau of Health, Dept. of Human Services, Augusta.

13. Hoxie, D.C., 1966: Radon-222 Determinations in Groundwaters. Masters Thesis, Rutgers, The State University of N.J.

14. Lundin, F.E., Jr., J.W. Lloyd, E.M. Smith, V.E. Archer, and D.A. Holaday, 1969: Mortality of uranium miners in relation to radiation exposure, hardrock mining and cigarette smoking - 1950 through Sept. 1967. Health Physics, 16:571-578.

15. Mantel, N. and W. Haenszel, 1959: Statistical aspects of the analysis of data from retrospective studies of disease. J. Nat. Cancer Inst., 22:719-748.

16. NCRP Report No.77, 1984: Exposures from the Uranium Series with Emphasis on Radon and its Daughters. National Council on Radiation Protection and Measurements. Bethesda, Maryland, p. 15.

17. NCRP Report No.78, 1984: Evaluation of Occupational and Environmental Exposures to Radon and Radon Daughters in the United States. National Council on Radiation Protection and Measurements. Bethesda, Maryland, pp. 7-10, 111.

18. National Research Council, Advisory Comittee on the Biological Effects of Ionizing Radiations, 1980: The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. National Academy of Sciences, Washington, D.C., p. 390.

19. Pershagen, G., L. Damber and R. Falk, 1984: Exposure to radon in dwellings and lung cancer: A pilot study. In: Proceedings of the Third International Conference on Indoor Air Quality and Climate, Vol. 2. Stockholm, Sweden, pp. 73-78.

20. Pohl, E. and J. Pohl-Ruling, 1980: Dose distribution in the human organism due to incorporation of radon and decay products as a base for epidemiological studies. In: Proceedings of the Specialist Meeting on the Assessment of Radon and Daughter Exposure and Related Biological Effects. G.F. Clementi, et al., eds. RD Press, University of Utah, Salt Lake City, pp. 210-219.

21. St. Clair Renard, K.G., 1974: Respiratory cancer mortality in an iron ore mine in Northern Sweden. Ambio, 3:67-69.

22. Sevc, J., E. Kung and V.P. Placek, 1976: Lung cancer in uranium miners and long term exposure to radon daughter products. Health Physics, 30:433.

23. Thompson, J.J., 1902: Phil. Mag., 4:352.

24. Trosko, J.E. and Chia-Cheng Chang, 1978: Environmental carcinogenesis: An integrative model. The Quarterly Review of Biology, 53:115-135.

25. Upton, A.C., 1982: The biological effects of low-level ionizing radiation. Sci. Amer., 246:41-49.

26. Weiffenbach, C.V., 1982: Radon, Water, and Air Pollution: Risks and Control. Land and Water Resources Center, University of Maine, Orono, p. 4.

APPENDIX

Maine Cooperative Radon/Health Project
Human Research Subjects Informed Consent

I understand that I am being asked to enter a research project to explore possible health effects of radon in the domestic environment. Radon is a naturally-occurring radioactive gas produced in bedrock which may find its way into households directly or by way of water supplied from groundwater wells. Although this project may continue for several years, I understand that my participation in it will be limited to the following activities, all of which should be completed within four months:

1. answering a questionnaire about my home, past occupations, smoking habits and health;

2. allowing a research team to come to my home at my convenience to obtain a sample of my drinking water and to leave a small air-monitoring cup which may remain in my home for two to four months; and

3. allowing the principal investigator (Dr. Peter W. Rand) to review my hospital records, if appropriate and when available.

There are no foreseen risks associated with this project. Its benefits to me are that I will eventually receive, free of charge, the results of radon measurements done on my water and home air; and the benefit to society may be information which may be useful in the prevention of disease.

I understand that I enter this project voluntarily and that I may withdraw from it at any time without loss of benefits to which I might otherwise be entitled; that I will not be charged for any aspect of this research; that my name will not be used in any publication resulting from this research; that all records relative to this research will be treated in confidence, being available only to the investigators or to agents of the federal government who oversee informed consent assurance; and that I am free to ask Dr. Rand (871-2163) any questions I might have relating to this project.

____
(Signature of Person Obtaining Consent)

____
(Patient or Legal Representative)

____
(Date)

____
(For Subjects Under 18 Years of Age)

____
(Parent or Guardian)

A copy of this consent form must be given each patient entering the study.

____
(Witness)

(Signature of witness necessary only when person signing form is other than the patient)

MAINE RADON PROJECT QUESTIONNAIRE

Please first take time to read this questionnaire through. The questions should be self-explanatory. Please answer as many as you can, leaving the others blank. Return the questionnaire with the signed consent form. After we have reviewed it, we will call you if we need more information.

If you are completing this questionnaire for someone else, please provide your name and your relationship to the patient:

Name of person filling out questionnaire______________________________

Relationship to patient:__________________________

PATIENT:

Name______________________________ Age__________
Date of birth_______/_____/______ Sex__________
Todays date________________________

Address____
____
__________________________________zip__________

Home Phone____

Family Doctor____
Address ____
___________________________zip__________

Have you been hospitalized in the last year? (Please circle) Yes No
(If answer is yes)
Name of hospital_______________________
Attending physician____________________
Approximate date of admission__________

EDUCATION

______years of grade school
______years of high school
______years of vocational/trade school
______years of college
______years of postgraduate education

HOUSING INFORMATION

Location: street/road________________________town__________________

ON THE ATTACHED MAP, PUT AN X WHERE YOUR HOUSE IS

How long have you lived in this house? from__________to__________
(year) (year)

How many rooms are there in this house?________
(Please do not count bathrooms, basements, and unoccupied attics)

House construction (Please check the most correct answer)
______woodframe
______brick/concrete
______stone (type if known)____________________
______mobile home
______other (please indicate)___________________

Is there exposed rock (other than in the foundation), such as a stone fireplace, anywhere in your house?
______yes (if yes, identify)___________________
______no

Does your house have a basement? (circle correct answer) yes no

Is the **foundation**:	Is the **floor**:
______concrete/cinderblock	______concrete
______rock (type__________)	______dirt
______other (____________)	______rock (type________________)

Do you work in or sleep in the basement? yes no

If yes, on the average, how much time do you spend in the basement? (Please indicate the average number of hours per week______and for how many years______.

If your house has no basement, is the house on: (please check best answer)
______posts ______ledge ______a slab

Over the last 10 years, has your house been: (Please check best answer)
______well insulated ______a little drafty
______very drafty ______don't know

What have been the principal sources of heat in your home for the last 10 years? (Please rank in order of importance 1, 2, 3, etc.)
______wood ______coal
______gas ______oil
______electricity ______solar

WATER USE INFORMATION

Company who drilled your well (if known)____________________________
Address________________________________
____

In what year was your well drilled?__________
well depth in feet______ average flow (gpm)______
length of well casing______

If you do not know this information about your well, please, (if possible), give name, address, and phone number of person who might know.

Name________________________________
Address_____________________________
____
Phone number________________________

Does your well ever run dry? (please check)
______never ______only in droughts
______frequently

Is there a water filter on the well? (please circle) yes no
If yes, year the filter was installed__________
Type of filter_________________________________

Since you have been using this well, please indicate the average number of:
______months of the year you have lived in this house.
______hours of the day you have spent out of the house.
______people who have lived in this house.
______total number of showers taken weekly by all members of household.

PREVIOUS WATER SUPPLIES:

Please fill out the form below to the best of your memory, going back as far as you can from the time the well for your present house was drilled, or starting with your previous house if your present house had a drilled well when you moved into it.

Town and State of Residence	Approximate Dates of Residency (19___ to 19___)	Water Source (check one): drilled well	dug well, spring, well point	lake, stream, pond	town water	other (specify)
1)						
2)						
3)						
4)						
5)						
6)						

MEDICAL HISTORY

List the condition(s) you are now being treated for and the year they were first diagnosed:

Condition	Year
______________________________	______________
______________________________	______________
______________________________	______________
______________________________	______________

Please check the following diseases for which you have been diagnosed **while you have been in this house** by putting the year of diagnosis next to the condition:

______high blood pressure
______heart disease
______blood vessel disease
______stroke
______other neurological disese
______diabetes
______gallstones
______kidney disease
______kidney stones
______bladder disease
______cirrhosis of the liver

______prostate trouble
______chronic bronchitis/emphysema
______allergies, hayfever/asthma
______arthritis
______gastric/duondenal ulcers
______diverticulosis
______rectal/colon polyps
______thyroid condition
______hepatitis
______cancer (type__________)
______other (type___________)
______other (type___________)

How many children have you had that were:		Of these, number born while you were living in this house:
born alive	______	______
number of miscarriages	______	______
number of stillborn	______	______
number of children with birth defects	______	______

Diagnostic Radiation

About how many x-rays or fluoroscopic exams have you had during your life? (do not include dental x-rays or x-rays for your present condition)

______0-10 ______10-20 ______20+

If many x-rays were required for any particular condition(s), please give approximate number________, area examined__________________________, and date___________________.

Radiation Treatment

Have you ever been treated with radium, x-rays, or radioactive isotopes?
(please circle) yes no
If yes, when_________(year)
For what disease?___________________
What part of the body?______________

FAMILY HISTORY

If known, in what country was your mother born?____________father?__________

Do you have a spouse that has lived in this house? ______yes ______no
Years: 19______ to 19______ Date of birth_____/_____/______

List any chronic or serious conditions he or she may have developed during this time:

Condition	Year Diagnosed

List any chronic or serious health problems suffered by your children who lived in your house when it was served by its present water supply:

Condition	Year Diagnosed	Years in this residence	Birthdate	Sex (M/ F)

SMOKING HISTORY

Cigarettes

Have you smoked at least one cigarette per day for one year? (please circle)
yes no
How old were you when you started?______(age)
Do you still smoke? (please circle) yes no
If no, at what age did you quit? __________(age)

If you stopped and started again, for how many years did you stop in all________(years)

Over the years, what is the average number of cigarettes you smoked per day?________(number)

Pipes

Have you smoked a pipe for more than a year at any time?
(please circle) yes no
How old were you when you started?______(age)
Do you still smoke? (please circle) yes no
If no, at what age did you quit?________(age)

If you stopped and started again, for how many years did you stop in all?_______(years)

Over the years, what is the average number of pipefuls you smoked per day?______(number)

Cigars

Have you smoked cigars for more than a year at any time?
(please circle) yes no
How old were you when you started?______(age)
Do you still smoke? (please circle) yes no
If no, at what age did you quit?_______(age)

If you stopped and started again, for how many years did you stop in all?________(years)

Over the years, what is the average number of cigars you smoked per day?______(number)

Other Tobacco Smoke Exposure

Over the years, please indicate the extent to which you have been exposed to other peoples tobacco smoke:

______very little ______moderately _______greatly

OCCUPATIONAL HISTORY

What is your current occupation and what are your duties?

(Occupation)____
(Duties)____
How many years on this job?__________(years)

If retired, what was your last occupation and what were your specific duties?
(Occupation)____
(Duties)____
How many years on this job?__________(years)

Please indicate the job, other than the one listed above, that you have held for the longest period of time:
(Occupation)____
(Duties)____
How many years on this job?__________(years)

In your work or daily life, are (were) you regularly exposed to any of the compounds listed below? If yes, please respond by indicating the number of years exposed, degree of exposure, your specific job, and your place of employment. Do not forget time spent in the service, wartime employment, or exposures associated with hobbies or recreation.

Compound	Exposed: Yes	Exposed: No	Years Exposed (19__ to 19__)	Degree Of Exposure: (S=slight; M=moderate; E=extreme)	Specific Job	Place Of Employment
asbestos	___	___	___	___	___	___
caustic chemicals/ acids	___	___	___	___	___	___
solvents, degreasers, plasticizers	___	___	___	___	___	___
coal/stone dust	___	___	___	___	___	___
coal tar/pitch/ asphalt	___	___	___	___	___	___
gasoline exhaust	___	___	___	___	___	___
diesel exhaust	___	___	___	___	___	___
dyes	___	___	___	___	___	___
formaldehyde	___	___	___	___	___	___
pesticides/ herbicides	___	___	___	___	___	___
textile fibers/ dust	___	___	___	___	___	___
wood dust	___	___	___	___	___	___
x-rays/ radioactive materials	___	___	___	___	___	___